RESEARCH ARTICLE

pISSN: 0126-074X | eISSN: 2338-6223
MKB. 2021;53(2):87–95
https://doi.org/10.15395/mkb.v53n2.2364

Correlation between Head Computed Tomography Scan, Pre-operative Blood Lactate, and Pre-operative Glucose Level in Acute Traumatic Subdural Hematoma

Jefri Henky,[1] Ahmad Faried[2]
[1]Department of Neurosurgery, Faculty Medicine of Baiturrahmah University/Semen Padang Hospital Padang, Indonesia, [2]Department of Neurosurgery Faculty of Medicine Universitas Padjadjaran/ Dr. Hasan Sadikin General Hospital Bandung, Indonesia

Abstract

Acute traumatic subdural hematoma (SDH) is a focal brain injury resulting in alteration of cerebral perfusion and glucose metabolism, which would also results in hyperglicemia-induced-hyperlactatemia. A cross-sectional study was performed to analyze acute traumatic SDH patients by head CT scan and observe the effect on pre-operative blood lactate and blood glucose levels in 40 acute traumatic SDH patients at Dr. Hasan Sadikin Hospital Bandung, Indonesia during the period of July–September 2013. Somers' D correlation were used in the analysis with a p-value of ≤0.05 considered as significant with 95% confidence interval. The mean values of pre-operative blood lactate and blood glucose levels were 3.16±1.49 mmol/L and 155.85±32.95 mg/dL, respectively with a strong positive correlation between the hematoma thickness and the increase in blood lactate (r=0.656; p=0.021) and a moderate positive correlation with increased blood glucose (r=0.556; p=0.025). In addition, the compressed cistern also had a very weak positive correlation with increase in blood lactate (r=0.156; p=0.043) and very weak positive correlation with increase in blood glucose (r=0.139; p=0.056) while the midline shift had a weak positive correlation with increased blood lactate (r=0.353; p=0.041) and a weak positive correlation with increased blood glucose (r=0.333; p=0.046). In conclusion, increased hematoma thickness, compressed cistern, and midline shift seen on head CT scan correlate with increasing blood lactate and glucose levels in acute traumatic SDH. Head CT scan, blood lactate level, and blood glucose level can be considered as one of the routine examinations to determine acute traumatic SDH severity at the macroscopic and cellular level.

Keywords: Acute traumatic subdural hematoma, blood lactate, blood glucose, head computer tomography scan

Hubungan CT Scan Kepala dengan Laktat Darah Preoperatif dan Kadar Glukosa pada Hematoma Subdural Traumatik Akut

Abstrak

Hematoma subdural traumatik akut (SDH), cedera otak fokal yang mengakibatkan perubahan perfusi otak dan metabolisme glukosa; mekanisme ini menyebabkan hiperglikemia-hiperlaktatemia. Studi potong lintang dilakukan untuk menganalisis pasien SDH traumatik akut yang dilakukan pemeriksaan *CT scan* kepala serta mengobservasi hubungannya dengan kadar laktat dan kadar glukosa darah pre-operasi pada 40 pasien SDH traumatik akut di Rumah Sakit Dr. Hasan Sadikin Bandung, Juli–September 2013. Menggunakan Somers' D dan p-value ≤0.05 dianggap signifikan dengan CI 95%. Rerata kadar laktat darah pre-operasi 3.16±1.49 mmol/L dan glukosa darah pre-operasi 155.85±32.95 mg/dL. Uji korelasi Somers' D menunjukkan ketebalan hematoma berkorelasi positif kuat dengan peningkatan laktat darah (r=0.656; p=0.021) dan berkorelasi positif sedang dengan peningkatan glukosa darah (r=0,556; p=0,025), kompresi sisterna berkorelasi positif sangat lemah dengan peningkatan laktat darah (r=0.156; p=0.043) dan korelasi positif sangat lemah dengan peningkatan glukosa darah (r=0.139; p=0.056), pergeseran garis tengah memiliki korelasi positif lemah dengan peningkatan laktat darah (r=0.353; p=0.041) dan korelasi positif lemah dengan peningkatan glukosa darah (r=0.333; p=0.046). Peningkatan ketebalan SDH, kompresi sisterna dan pergeseran garis tengah berkorelasi dengan peningkatan kadar laktat dan glukosa darah pada SDH traumatik akut. *CT scan* kepala, laktat darah dan glukosa darah dijadikan salah satu pemeriksaan rutin menentukan keparahan SDH traumatik akut pada tingkat makroskopik dan sel.

Kata kunci: Hematoma subdural traumatis akut, laktat darah, glukosa darah, pemindaian tomografi komputer kepala

Corresponding Author: Jefri Henky, Department of Neurosurgery, Faculty Medicine of Baiturrahmah University/Semen Padang Hospital Padang, Jl. By Pass KM 15, Kota Padang, West Sumatra, Indonesia,, Email: neurosurg_henky@yahoo.co.id

Introduction

Traumatic brain injury (TBI) is one of the causes of death and disability in humans at the productive age of ≤ 45 years. Based on the International Mission for Prognosis and Clinical Trial (IMPACT)data; death and disability at that age range was dominated by male with a ratio of 3: 1. Studies suggest that the incidence of TBI is between 18 and 250 per 100,000 persons per year. Men and people living in social and economical deprived areas, usually young adults and the elderly are high-risk groups for TBI.[1]
In the implementation of health services, TBI challenges neurosurgeons to always work better in order to reduce the disability rate which is approximately 25-35% and the mortality rate is quite high. In addition, the adequacy of hospital care facilities and the development of drugs will help the recovery process of post-TBIpatients. The true burden of TBI and its sequelae appears to be underestimated owing to incomplete capture of data, especially in low- and middLe-income countries (LMICs).[2] Nevertheless, LMICs face almost three times more cases of TBI proportionally than high-income countries (HICs).

One of the focal brain injuries which cause neurological deficits with sequelae and even death after TBI is acute traumatic SDH. Based on the Traumatic Coma Data Bank (TDCB), the incidence of traumatic SDH was about 24% of all TBI incidents, with 21% manifesting as severe head injury, 11% as minor head injury and 68% as moderate head injury. The mortality rate among all intracranial focal lesions is quite high, approximately 50-60% with several outcome predictive factors, such as age, duration of occurrence, accompanying trauma, the Glasgow coma scale (GCS) value, thickness of hematoma, narrow cisterns, midLine shift, brain edema/ contusions and the presence of subarachnoid bleeding (SAB).[3,4]

The pathogenesis of cellular injury due to acute traumatic SDH is the duration of congestion of the superficial veins in cerebral hemispheres and the extraordinary activation of excitatory neurotransmitter receptors and glutamatergic N-methyl-D-aspartate (NMDA) receptors which can cause neuronal damage such as ischemic necrosis; ischemic occurs due to thin compression of the hematoma (the hematoma that in the shape of a thin crescent) or for a short period of time or due to the contusions and edema it causes.[4,5]

Under extreme conditions such as acute traumatic SDH, aerobic metabolism is compromised by decreased oxygen and glucose supply, mitochondrial damage or increased release of excitatory neurotransmitters resulting in increased intracellular lactate accumulation. Lactate production can occur in all body tissues, such as skeletal muscle, brain, red blood cells and kidneys. The level of lactate in normal humans can decline rapidLy with an average rate of 320 mmol / L / hr. This decline is a result of metabolism by the liver and the process of lactate reconstruction back to pyruvate. The lactate level is maintained in a basal state <1 mmol/L in venous and arterial blood.[6]

Delivery of glucose to the brain is facilitated by the glucose transporter 3 (GLUT 3) which is independent of insulin action but is dependent on blood delivery or adequate perfusion. In acute traumatic SDH and brain ischemia, glucose transporters and glucose metabolism processes are disrupted and there is an increase of lactate uptake by neurons in the brain due to increased energy requirements resulting in hyperglicemia-induced-hyperlactatemia. This process will cause a systemic hyperglycemic reaction as a result of high levels of lactate due to hyperglycolysis in the brain tissue and hypoglycemia in the brain tissue.[6–8]

On head computer tomography (CT) scan, an acute traumatic SDH is described as hyperdense, crescent-shaped lesion which lies between the dura mater and the parenchyma of the brain. They are often surrounded by hypodense lesions which are edematous lesions in the brain parenchyma, with narrowing of the groove and compression of the gyrus in the cerebral cortex. These conditions result in a shift in the midLine or midLine shift and a narrowing of the cisterns. The head CT scan is clinically associated with the outcome of acute traumatic SDH and is also used as an indication for surgery.[9]

Head CT scan indicative of acute traumatic SDH surgery are characterized by hematoma thickness >10 mm and hematoma volume, midLine shift>5 mm and patency of the basal cistern. Previous studies showed that patients with old age, midLine shift>5 mm, and emphasis on basal cisterns correlated with mortality of >70–80%. Patients hematoma thickness <10 mm were reported to have a mortality rate of 10% whereas patients with hematoma thickness >30 mm had a reported mortality rate of 90%.[9] Furthermore, time from TBI to surgery may not be as important as time from clinical deterioration or onset of cerebral herniation to surgery. The literature supports the statement

that the length of time from clinical deterioration to operative treatment of an SDH is significantly related to outcome.

Methods

The current study is an observational analytic study with cross sectional design aimed to analyze acute traumatic SDH patients by head CT scan and observing their effect on pre-operative blood lactate and blood glucose levels. This study was conducted in Dr. Hasan Sadikin Hospital (RSHS), Bandung, July-September 2013 with Ethical Committee approval number 273/UN6.C2.1.2/KEPK/PN/2013, 40 patients was achieved based on the correlation analysis formula. The subjects of this study are adult acute traumatic SDH patients (≥ 18 years) who met the indication for surgery whose families gave the permission to participate in the study. Blood lactate and blood glucose levels from the patients are measured prior to acute traumatic SDH.

Exclusion criteria for this study are acute traumatic SDH patients whose injuries are accompanied by focal extradural lesions such as epidural hematoma (EDH), intradural focal lesions such as intracerebral hematoma (ICH), diffuse axonal injury lesions, multiple extradural injuries, failure or disturbance of respiration and systemic circulation since the onset of trauma, systemic disease before trauma, such as diabetes, liver, lung, heart, kidney disease and are known to receive conservative therapy from other hospitals by administering osmotic diuretics and corticosteroids. The independent variables of this study were SDH thickness, cistern compression and midLine shift as seen on head CT scan, meanwhile the dependent variables were blood lactate levels and blood glucose levels obtained from blood tests of acute traumatic SDH patients.

Data were analyzed using the SPSS for windows ver. 20. Data of subject characteristics are displayed descriptively and data distribution will be assessed using the Kolmogorov Smirnov test. Somers' D correlation test was used to test the correlation of preoperative lactate levels and blood glucose levels with each independent variable. The significance is determined based on Table 1, regarding the reference value for correlation (r), significance (p) and direction of correlation. The set value of confidence interval (CI) in this study was 95%.

Results

In this study, there were 40 acute traumatic SDH patients with the mean age of 44.73±19.76 years. The percentage male patients were 62.5% with the male to female ratio of 2: 1. The mean operating time interval in this study was 17.08±11.86 hours. Approximately 67.5% of acute traumatic SDH patients were admitted to the hospital with moderate head injury (GCS 9-13), followed by severe head injury (GCS 3-8) in 30% and minor head injury (GCS 14-15) in 2.5% (Table 2).

Based on head CT scan, 50% of patients with acute traumatic SDH had a hematoma thickness <15 mm and 50% had a thickness of 15-20 mm. Approximately 62.5% showed emphasis on the basal cistern and 80% with a midLine shift image

Table 1 Interpretation of Correlation Test Results Based on Correlation Strength (r). Significance (p). and Correlation Direction

Parameter	Value	Interpretation
Correlation Strength (r)	0.0 to <0.2	Very weak
	0.2 to <0.4	Weak
	0.4 to <0.6	Medium
	0.6 to <0.8	Strong
	0.8 to 1	Very strong
Significance (p)	p<0.05	There is a significant correlation between the two variables tested
	p>0.05	There was no significant correlation between the two variables tested
Correlation Direction	+ (positive)	Unidirectional. the greater the value of one variable. the greater the value of the other variables
	- (negative)	In opposite directions. the greater the value of one variable. the smaller the value of the other variables

Table 2 Patients Characteristics

Characteristics	Statistical Value
Age	44.73±19.76 (18–82)
Sex	
Male	25/40 (62.50%)
Female	15/40 (37.50%)
Surgical interval (hours	17.08±11.86 (2–48)
Glasgow coma scale during admission	
Mild head injury (GCS 14–15)	1/40 (2.50%)
Moderate head injury (GCS 9–13)	27/40 (67.50%)
Severe head injury(GCS 3–8)	12/40 (30.00%)
SDH thicknessaccording tohead CT scan	
<15 mm	20/40 (50.00%)
15–20 mm	20/40 (50.00%)
Cistern compressionaccording tohead CT scan	
Compressed	25/40 (62.50%)
Not Compressed	15/40 (37.50%)
MidLine shift according tohead CT scan	
>5 mm	32/40 (80.00%)
<5 mm	8/40 (20.00%)
Preoperative hemoglobin level (g/dL)	12.47±1.71 (9.20–15.40)
Preoperative blood lactate level (mmol/L)	3.16±1.49 (1.00–6.60)
Preoperative blood glucose level (mg/dL)	155.85±32.95 (107–223)

>5 mm (Table 2).

The laboratory results are as follows: the mean preoperative hemoglobin (Hb) level was 12.47±1.71 g/dL, the mean pre-operative blood lactate level in acute traumatic SDH patients was 3.16±1.49 mmol/L and the mean level of pre-operative blood glucose in acute traumatic SDH patients was 155.85±32.95 mg/dL. The data normality test was carried out on all numerical variables in Table 2.

In this study, almost all variables had a *p value*>0.05, except for the operating time interval variable which was not normally distributed with a *p value*<0.0001. The Kolmogorov Smirnov test showed that the variable age is normally distributed (p=0.098), the preoperative Hb level variable is normally distributed (p=0.163), the pre-operative blood lactate level variable is normally distributed (p=0.158) and the pre-operative blood glucose level variable is also normally distributed (p=0.055).

The SDH thickness correlation in Somers' D correlation test to preoperative blood lactate levels was significant (p<0.05) with a correlation coefficient of 0.656 which indicated that there was a strong positive correlation between SDH

Table 3 Correlation of SDH Thickness Based on Head CT Scan with Preoperative Blood Lactate and Blood Glucose Levels

Variable		SDH thickness		Total	Correlation (r)	Sig. (p)
		<15mm	15-20 mm			
Pre-operative blood lactate level	Normal	3	5	8	0.656	0.021
	Hyperlactatemia	17	15	32		
Pre-operative blood glucose level	Normal	4	0	4	0.556	0.025
	Hyperglycemia	16	20	36		

* Somers'd Correlation Test

Table 4 Correlation of Cisternal Compression Based on Head CT Scan with Preoperative Blood Lactate and Blood Glucose Levels

Variabel Compressed		Cistern compression		Total	Correlation (r)	Sig. (p)
		Not compressed				
Pre-operative blood lactate level	Normal	4	4	8	0.156	0.043
	Hyperlactatemia	21	11	32		
Pre-operative blood glucose level	Normal	2	2	4	0.139	0.056
	Hyperglycemia	23	13	36		

* Somers'd Correlation Test

Table 5 Midline Shift Correlation Based on Head CT Scan with Pre-operative Blood Lactate and Blood Glucose Levels

Variabel >5 mm		Midline Shift		Total	Correlation (r)	Sig. (p)
		<5 mm				
Pre-operative blood lactate level	Normal	6	2	8	0.353	0.041
	Hyperlactatemia	26	6	32		
Pre-operative blood glucose level	Normal	2	2	4	0.333	0.046
	Hyperglycemia	30	6	36		

* Somers'd Correlation Test

thickness and preoperative blood lactate levels. The correlation of SDH thickness to preoperative blood glucose levels was also significant (p<0.05) with a correlation value of 0.556 which indicated a moderate positive correlation between SDH thickness and preoperative blood glucose levels (Table 3).

The correlation of cistern compression in the Somers' D correlation test to pre-operative blood lactate levels was significant (p<0.05) with a correlation value of 0.156 which indicated a very weak positive correlation between cistern compression and pre-operative blood lactate levels.The correlation of cistern compression to pre-operative blood glucose levels was not significant (p>0.05) with a correlation value of 0.139 which indicated a very weak positive correlation between cisterncompression and pre-operative blood glucose levels (Table 4).

MidLine shift correlation in Somers' D correlation test to preoperative blood lactate levels was significant (p<0.05) with a correlation value of 0.353 which indicated a weak positive correlation between midLine shift and preoperative blood lactate levels. The midLine shift correlation to preoperative blood glucose levels was also significant (p<0.05) with a correlation value of 0.333 which indicated a weak positive correlation between midLine shift and preoperative blood glucose levels (Table 5).

The correlation of preoperative blood lactate levels in the Somers' D correlation test to preoperative blood glucose levels was significant (p<0.05) with a correlation value of

Table 6 Correlation of Pre-operative Blood Lactate Levels with Preoperative Blood Glucose Levels

Variabel Normal		Pre-operative Blood Lactate Level		Total	Correlation (r)	Sig. (p)
		Hyperlactatemia				
Pre-operative blood glucose level	Normal	1	3	4	0.568	0.037
	Hyperglycemia	7	29	36		

* Somers'd Correlation Test

0.568 indicated a moderate positive correlation between pre-operative blood lactate levels with pre-operative blood glucose levels (Table 6).

Discussion

The mean age of acute traumatic SDH patients in this study is in accordance with the TBI epidemiological data reported by IMPACT in the previous literature which stated that TBI is one of the causes of death and disability in productive age populations(≤45 years). One of the contributing factors to this is the factthat productive age populationshave more outdoor activities such as work and sports which influenced the accident rate. Age of acute traumatic SDH patients is one of the factors influencing the outcome.It is stated in the literature that patients with older age has 4 times the mortality rate or worse outcome than patients with younger age. [1,4,10,11]

In this study, the male gender was about 62.5% with a ratio of 2:1 male to female, this ratio is slightly lower than the literature which states that TBI is more often found in the male group 3: 1 in women. This ratio occurs because we thought that more male activities outside the home with heavy work and using vehicles than women[1,12].

Approximately 67.5% of acute traumatic SDH patients admitted with moderate head injury (GCS 9-13), followed by severe head injury (GCS 3–8) in 30% and minor head injury (GCS 14–15) in 2.5%. This is almost the same as the data held by TDCB, that 68% of patients with acute traumatic SDH manifested as moderate head injury, 21% manifested as severe head injury and 11% manifested as minor head injury. The GCS value has a strong relationship with the outcome and recovery of patient neurological function.[6,7,11]

The surgery time interval is also a confounding variable that is taken into account in this study. It was found that the average operating time interval was 17.08 + 11.86 hours. The average surgery time interval of this study is >8 hours, more than the golden hour,but still not past the silver day (<24 hours).[13]

On the CT scan of the head, there were 50% with a hematoma thickness <15 mm and 50% with a hematoma thickness of 15–20 mm; whereas for other variables, about 62.5% with emphasis on basal cisterns and 37.5% without basal cisterns emphasis. The results of this study can be interpreted that the hematoma thickness of acute traumatic SDH can cause a focal effect on brain tissue in the form of brain superficial veins congestion and impaired brain perfusion so that it can progress to cerebral edema which results in compression of the basal cistern which indicates an elevated ICP. [4,9,11,14]

The focal effect of the hematoma and cerebral edema can be seen in this study in the form of a midLine shift >5 mm, which is 80% and a midLine shift <5 mm is 20%. According to several studies, all aspects of the CT scan of the head are indirect factors that determine the outcome of patients with acute traumatic SDH. [4,8,9,14]

From blood laboratory examination, the mean preoperative hemoglobin level in this study was 12.47 + 1.71 g/dL. Mean hemoglobin levels in this study were still within normal limits, with a reference value of 12.1–17.6 g/dL for male patients and 11.7–16.2 g/dL for female patients.[15]

The mean preoperative blood lactate level for acute traumatic SDH patients was 3.16 + 1.49 mmol/L. The mean value of blood lactate levels in this study exceeded the normal limit of blood lactate levels in adult patients, namely 0.9-1.7 mmol/L. The elevation in blood lactate levels is in accordance with the results of studies reported by Kawamata et al.[13] and Cureton et al. [6] that blood lactate levels could increase shortly after TBI and increase about 23.7% from baseline, then would be normal in 24-36 hours if the annoyance and damage was olved.

In addition, the mean preoperative blood glucose level in patients with acute traumatic SDH was 155.85 + 32.95 mg/dL. The mean value of blood glucose levels in this study exceeds the normal limit of adult patients, namely 70–115 mg/dL. This increase in blood glucose levels is in accordance with research conducted by Magnoni et al. that systemic hyperglycemia was a response to glucose metabolic disorders and a response to increased levels of lactate in brain tissue.[6–8,15]

The results of the correlation analysis between GCS at the time of hospitalization and preoperative blood lactate levels showed a very weak negative correlation. Likewise, the results of the GCS correlation analysis on admission to the preoperative blood glucose levels showed a very weak negative correlation. The results of this correlation reinforce the assumption that the smaller the GCS value or the worse the awareness of the acute traumatic SDH patient, the higher the blood lactate level and the blood glucose level of the acute traumatic SDH patient, so that the GCS of the acute traumatic SDH patient can reflect an increase in blood lactate levels and blood glucose levels; due to hyperglycolysis and

glucose metabolic disorders that occur. [5,7,8,17,18]

The next correlation analysis was the operation time interval with preoperative blood lactate levels which showed a very weak positive correlation. Meanwhile, the correlation analysis of the operation time interval with the preoperative blood glucose level also showed a weak positive correlation. This means, the longer the operation time interval, the higher the blood lactate level and blood glucose level of acute traumatic SDH patients, because the length of the operation time interval in acute traumatic SDH patients illustrates the length of focal damage to the brain, the duration of impaired brain tissue perfusion, the duration of cerebral edema. that occurs and the duration of glucose metabolic disorders and hyperglycolysis that occurs so as to produce large amounts of lactate. [6–8,17] The correlation of SDH hematoma thickness and preoperative blood lactate levels showed a strong positive correlation. Meanwhile, the correlation between SDH thickness and preoperative blood glucose levels showed a moderate (moderate) positive correlation. These results indicate that the thicker the SDH hematoma, the higher the blood lactate and blood glucose levels, because the thickness of the hematoma will reflect the magnitude of focal emphasis on the brain tissue of acute traumatic SDH patients. [4,5,9,11,14]

The correlation of cistern compression with preoperative blood lactate levels showed a very weak positive correlation. While the correlation of cistern compression with preoperative blood glucose levels was not significant and showed a very weak positive correlation. Although the correlation of cisternal compression with blood lactate levels and blood glucose levels is very weak and insignificant on blood glucose, the researchers understand that cistern compression is a continuation effect of focal suppression and brain edema that occurs over a long period of time. This reason is reinforced because 62.5% of cases of cisternal compression were comparable to 50% of cases of hematoma thickness of 15-20 mm, meaning that only a large proportion of study subjects had severe brain edema that compressed the basal cisterns. The edema can be seen its effect on the increase in blood lactate levels and blood glucose levels in acute traumatic SDH patients.[4,5,9,11,14]

Regarding the focal suppression of hematoma and brain edema discussed above, it appears that the midLine shift correlation with preoperative blood lactate levels shows a weak positive correlation and the midLine shift correlation with preoperative blood glucose levels also shows a weak positive correlation.). The results of this analysis mean that the greater the midLine shift that appears on the CT scan of the head of a acute traumatic SDH patient, the higher the blood lactate level and blood glucose level of the patient. This is a combination of the effects of hematoma suppression and brain edema, which aggravates hyperglycolysis and aggravates impaired glucose metabolism in brain tissue after acute traumatic SDH..[4,5,9,11,14]

The results of blood laboratory tests that were also considered as a confounding variable were hemoglobin levels, the correlation between hemoglobin levels and preoperative blood lactate levels showed a very weak negative correlation. Meanwhile, the correlation between preoperative hemoglobin levels and preoperative blood glucose levels shows a weak negative correlation. The correlation between these two variables means that the lower the hemoglobin level (anemia), the higher the blood lactate and blood glucose levels. This correlation is not a causal relationship due to the effect of hematoma suppression or brain edema that occurs, but it means that hemoglobin disruption will reduce oxygen and glucose supply to brain tissue so that it can interfere with glucose metabolism and glycolysis.[6–8,17]

The correlation of preoperative blood lactate levels with preoperative blood glucose levels showed a moderate positive correlation between preoperative blood lactate levels and preoperative blood glucose levels. These results indicate that the higher the blood lactate level of the acute traumatic SDH patient, the higher the blood glucose level of the acute traumatic SDH patient and vice versa.

If associated with the mechanism of increased lactate due to acute traumatic SDH, this correlation analysis is acceptable because the pathophysiology of hyperlactatemia due to hyperglycolysis and impaired glucose metabolism in brain tissue will systemically induce glucose. This response is known in the literature as hyperglicemia-induced-hyperlactatemia and it was proven biochemically and statistically in this study of acute traumatic SDH patients.[19,20]

In this study, not all variables were analyzed due to the limited number of samples and time of the study, so it was realized that there were many biased factors that influenced it. In addition, technical factors for blood sampling to check lactate and glucose levels must also be considered. In this study, all blood samples of acute traumatic SDH patients were taken from venous blood, because it was easier to obtain,

faster and considered ethical aspects of the patient.

The literature also proves that venous blood lactate has no significant difference with arterial blood lactate, however, the best blood sample for examining blood lactate levels is from a venous blood sample. While the blood sample for checking blood glucose levels should be from arterial blood samples, because the difference in venous blood glucose is estimated to be 10% higher than arterial blood glucose.[15]

Other bias factors have been identified from the inclusion criteria and exclusion criteria and attempted to be homogeneous and proven based on the normality test of the variables with the results of all variables being normally distributed. Therefore, this study results can be used as material for consideration, guidelines and references for the explanation of the patient's condition, patient treatment and outcome estimates for acute traumatic SDH patients. The results of this study recommend the examination of blood lactate levels and blood glucose levels as routine checks in acute traumatic SDH patients.

In conclusion, the thicker the SDH, the more compressed the cisterns and the greater the midLine shift on the CT scan of the head, the higher the blood lactate levels and blood glucose levels in acute traumatic SDH patients. CT scan of the head, blood lactate levels and blood glucose levels can be considered as routine tests in determining the macroscopic and cellular severity of acute traumatic SDH.

References

1. Popescu C, Anghelescu A, Daia C, Onose G. Actual data on epidemiological evolution and prevention endeavours regarding traumatic brain injury. J Med Life. 2015;8(3):272–7.
2. Hutchinson PJ, Kolias AG, Tajsic T, Adeleye A, Aklilu AT, Apriawan T, et al. Consensus statement from the International Consensus Meeting on the role of decompressive craniectomy in the management of traumatic brain injury consensus statement. Acta Neurochir (Wien). 2019;161(7):1261–74.
3. Robertson C, Castilla L. Critical care management of traumatic brain injury. In: Winn H, Berger M, Dacey R, editors. Youmans and Winn neurological surgery. Philadelphia: Elsevier Saunders; 2011. p. 3397–423.
4. Shahlaie K, Zweinenberg-Lee M, Muizelaar J. Clinical pathophysiology of traumatic brain injury. In: Winn H, Berger M, Dacey R, editors. Youmans and Winn Neurological Surgery. Philadelphia: Elsevier Saunders 2011. p. 3362–79.
5. Mckee AC, Daneshvar DH. The neuropathology of traumatic brain injury. Handb Clin Neurol. 2015;127:45–66.
6. Clausen F, Hillered L, Gustafsson J. Cerebral glucose metabolism after traumatic brain injury in the rat studied by 13C-glucose and microdialysis. Acta Neurochir (Wien). 2011;153:653–8.
7. Magnoni S, Tedesco C, Carbonara M, Pluderi M, Colombo A, Stocchetti N. Relationship between systemic glucose and cerebral glucose is preserved in patients with severe traumatic brain injury, but glucose delivery to the brain may become limited when oxidative metabolism is impaired: Implications for glycemic control. Crit Care Med. 2012;40(6):1785–91.
8. Meierhans R, Brandi G, Fasshauer M, Sommerfeld J, Schüpbach R, Béchir M, et al. Arterial lactate above 2 mM is associated with increased brain lactate and decreased brain glucose in patients with severe traumatic brain injury. Minerva Anestesiol. 2012;78(2):185–93.
9. Zacko J, Harris L, Bullock R. Surgical management of traumatic brain injury. In: Winn H, Burger M, Dacey R, editors. Youmans and Winn Neurological Surgery. Philadelphia: Elsevier Saunders; 2011. p. 3424–3252.
10. Leitgeb J, Mauritz W, Brazinova A, Janciak I, Majdan M, Wilbacher I, et al. Outcome after severe brain trauma due to acute subdural hematoma: Clinical article. J Neurosurg. 2012;117(2):324–33.
11. Sharma R, Rocha E, Pasi M, Lee H, Patel A, Singhal AB. Subdural hematoma: predictors of outcome and a score to guide surgical decision-making. J Stroke Cerebrovasc Dis. 2020;29(11):105180.
12. Bullock M, Hovda D. Introduction to traumatic brain injury. In: Winn H, Berger M, Dacey R, editors. Youmans and Winn neurological surgery. Philadelphia: Elsevier Saunders; 2011. p. 3267–9.
13. Grevfors N, Lindblad C, Nelson DW, Svensson M, Thelin EP, Rubenson Wahlin R. Delayed neurosurgical intervention in traumatic brain injury patients referred from primary hospitals is not associated with an unfavorable outcome. Front Neurol. 2021;11:610192.
14. Jiang C, Cao J, Williamson C, Farzaneh N,

Rajajee V, Gryak J, Najarian K, et al. Midline shift vs. mid-surface shift: correlation with outcome of traumatic brain injuries. Proceedings (IEEE Int Conf Bioinformatics Biomed). 2019;2019:1083–6.
15. Fischbach F, Dunning MB. A Manual of Laboratory and Diagnostic Tests. 9th ed. Lippincott Williams and Wilkins; 2015. p. 53–173.
16. Romeu-Mejia R, Giza CC, Goldman JT. Concussion pathophysiology and injury biomechanics. Curr Rev Musculoskelet Med. 2019;12(2):105–16.
17. Yu LY, Pei Y. Insulin neuroprotection and the mechanisms. Chin Med J (Engl). 2015;128(7):976–81.
18. Hansen T, Jensen T, Clausen B, et al. Natural RNA circles function as efficient microRNA sponges. Nature. 2013;495:384–8.
19. Zhou J, Burns MP, Huynh L, Villapol S, Taub DD, Saavedra JM, et al. Temporal changes in cortical and hippocampal expression of genes important for brain glucose metabolism following controlled cortical impact injury in mice. Front Endocrinol (Lausanne). 2017;8:231.
20. Brooks GA. The tortuous path of lactate shuttle discovery: From cinders and boards to the lab and ICU. J Sport Heal Sci. 2020;9(5):446–60.